

case, age 17, died three months from the first treatment from syphilitic meningitis. The Wassermann findings in the serum and spinal fluid did not change from a triple positive during the intervals. These cases might have resulted differently with the Swift-Ellis treatment. The third case is an epileptic, and so far all intravenous and mercurial treatment has not changed the degree of the intensity of the test or the number of convulsions. She has had nine convulsions a year for five years. She is now being treated with iodipin, and if we find the spinal fluid positive, Dr. Dawson will get the consent of the parents to give the Swift-Ellis treatment. At Agnews one case of hereditary syphilis is receiving the treatment, also a number of general paresis cases. Of thirty-eight cases of paresis in one institution the serum and spinal fluid both give triple positive in twenty-three cases. Two, an + positive serum and +++ positive spinal fluid. Three ++ positive serum and +++ positive spinal fluid. Ten cases gave a negative serum test with a triple positive spinal fluid. A few of these cases were checked by the Noguchi modification with the same results. Captain C. G. Snow of the General Letterman Hospital, Presidio, has checked a number and Dr. W. T. Cummins of the Southern Pacific Hospital a few of these cases, with the Lang's colloidal gold chloride test with some interesting findings. The spinal fluid of two meningitis cases, the patients showing mental symptoms, and giving a history of a recent infection, gave an absolute negative Wassermann with all other findings positive. That is, a differential cell count, a Nonne and butyric acid test. Both received prompt treatment with salvarsan intravenously and mercury with prompt and satisfactory return to normal. These were not institutional cases.

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PROSTATIC CARCINOMA IN A YOUTH.

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L. P. R. Age 17 years. American. Machinist apprentice. Admitted Oct. 2, 1911. Family history negative. Previous history—no recorded illness except diphtheria 9 years ago.

Present illness: Onset about seven weeks ago with colicky pains in lower left side of abdomen accompanied by vomiting. Pains radiated to left testicle. No hematuria. Incontinence of urine affected by posture. Three weeks ago pain shifted to right side of lower abdomen and radiated to right testicle. Has lost much weight.

Examination: Well nourished, fairly well developed young man. Pulse and respiration normal. Eyes normal. No eruption. Tongue clean. Heart and lungs negative. Rectal examination bimanually shows hard, lobulated enlargement of the prostate, which was particularly painful on palpation.

Urinalysis: Clear; sp. gr. 1018; acid; no sugar nor albumin; no casts.

Death occurred on Jan. 12, 1912.

Post mortem record. Markedly emaciated. Lividity and rigidity moderate. No eruption, scars, nor bed sores. Peritoneum smooth, moist, glistening and no increase of fluid. Position of abdominal organs normal. A large, firm, pale, nodulated mass completely fills pelvis. Spleen somewhat increased in size but of normal shape. Capsule strips with some difficulty. Cut surface shows markings of fibrosis. Color brownish red. No evidences of tumor metastasis. Liver of normal size and shape, and shows typical "nut-meg" markings. No evidences of tumor metastasis. Gall bladder, stomach, intestines and pancreas normal. Both kidneys somewhat larger than normal. Capsule strips easily. Color brownish red. Considerable dilatation of calyces as well as pelvis. Both ureters dilated throughout their continuity to size of lead pencil. Both adrenals soft, yellow and cystic.

The bladder is moderately dilated and contains approximately 200 cc. of urine. Its wall is considerably thickened and varies between 0.5 and 1 cm. The mucous membrane is moderately congested, thickened and rugose but no evidence of ulceration. In the prostatic region there lies a firm, pale, nodular mass which has exerted considerable pressure upon the rectum, as dilatation is seen above the area of stenosis. This mass includes the neck of the bladder and as far back as the openings of the ureters. Two small masses project into the bladder. On sectioning, these masses are firm and pale with whorls and bands of tissue apparently of connective type. There are no evidences of congestion nor hemorrhage. The prostatic urethra is stenosed and the mucous membrane of this as well as the membranous portion is moderately congested.

Mesenteric nodes are enlarged, firm and pale, their average size being about that of a soup bean. The retroperitoneal group are enlarged to the size of chestnuts with the same general consistency and appearance as the mesenteric group. Near the splenic flexure of the colon there is a pedunculated lymph node somewhat larger than a horse-chestnut (6x5x4 cm.). Its tissues are identical in appearance with the other nodes. Though very pale this was at first mistaken for an accessory spleen.

Permission for a partial autopsy only was granted so that an examination of the thoracic organs and central nervous system could not be made.

Histological examination: Spleen. Capsule and trabeculae show moderate fibrosis. Sinuses in places considerably dilated. Large quantities of hemosiderin are seen. Liver. There is no abnormality except for a moderate passive congestion and hemosiderosis of parenchyma near the midlobular areas. Kidneys. Capsule is moderately fibrosed. Much of the cortical epithelium shows degenerative and necrotic changes. In some of the tubules the epithelium has desquamated. There are numerous areas of connective tissue overgrowth. Adrenals. Marked vacuolation of the cells of the fascicular and reticular zones. Pancreas. A moderate fibrosis is evident. Prostate. There is considerable overgrowth of connective tissue. The epithelium of many alveoli shows marked proliferation and penetration of the basement membrane. In some places evidences of alveoli are seen but in many other places the epithelial masses are solidly formed. Retroperitoneal lymph nodes. All of these are of the same general structure. The connective tissue shows some overgrowth and between these trabeculae there are large and small masses of cells with vesicular nuclei resembling closely the above-mentioned alveolar epithelium of the prostate.

Clinical diagnosis: Sarcoma of the prostate.

Pathological diagnosis: Carcinoma of the prostate and retroperitoneal lymph nodes; chronic in-

terstitial splenitis and hemosiderosis; passive congestion of the liver; chronic parenchymatous nephritis; hydronephrosis and hydrometer; chronic interstitial pancreatitis.

THE RELATION OF LOCALIZED TENDERNESS TO THE SITE OF THE CAUSAL LESION IN PERFORATIVE PERITONITIS.*

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In keeping with this symposium the bearings of this paper will center on perforation is gastroduodenal disease. Four cases of perforation of ulcer of these parts coming within the writer's personal experience form the clinical basis of this paper.

The time limit will permit neither the consideration of the relations of the symptom under special view to the larger symptomatology of perforation, nor to the differential diagnosis.

The weight of present-day judgment seems to be that within several hours from the time of perforation there is usually a widespread, diffuse abdominal tenderness, and in addition "careful search will reveal an area of exquisite intensity overlying the ulcer."¹ Other localized areas of special tenderness with a single exception, are not dwelt upon, as one of the generally recognized occasionally associated features of the condition.

A number of authorities dwell upon the fact that with perforation in certain cases of duodenal ulcer, the main symptoms may become localized in the cecal region, and have often led to operation for appendicitis, instead of a first, direct surgical attack upon the upper digestive tract. The real source of these symptoms has even been overlooked after this misapplied surgery. Moynihan, as early as 1901, found 49 recorded cases of perforated duodenal ulcer resembling appendicitis, in 18 cases of which the first abdominal incision had been made over the appendix. His explanation is that the foreign fluid following the right-sided para-colic peritoneal planes, reaches the *caput coli* and causes there the local serous irritation which results in so much symptomatic confusion and surgical error. Even within the first three hours following perforation, greater abdominal resistance and more marked, even exquisite, tenderness may exist at the usual site of the appendix.

It may be that this was the exceptional condition Munro had in mind, when speaking broadly of peritonitis but without detailed reference or attempted explanation, stated that "the tenderness and spasm, with few exceptions, are located over the area of more marked infection."² Ordinarily, however, in perforation of both gastric and duodenal ulcers, the extruded fluid runs at large in the peritoneal cavity, in an indefinite way, producing diffuse peritonitis.

In view of what clinical experience has established in regard to local symptoms developing in

the right iliac region, does it seem unreasonable that similarly localized symptoms of irritation should arise in the presence of an advancing peritonitis in other portions of the abdomen as well? It has not, however, thus far come to the writer in his search, that except as already noted, other associated areas of special irritation are generally recognized. That, however, restricted irritative symptoms at a distance from the ulcer, amongst which localized tenderness must be one, are often enough met with but wrongly interpreted prior to the operation, is strongly suggested by the surgical errors reported in connection with operations in the course of peritonitis from gastroduodenal disease. Often enough, the operator's efforts for a short search and a quick operation are hampered by a misinterpretation of symptoms and a consequent disadvantageously placed abdominal incision. Yet in the long run, the amount of manipulation of the viscera, the operative trauma, the time consumed in operation affect decidedly the mortality rate.

The findings of the writer are at variance in some respects with what seems to be the generally accepted relationship of localized tenderness in this disease. The apparently controverting testimony noted in his cases, if admitted, may, therefore, be regarded as exceptional.

A possible source of difference between observers as to conclusions regarding sensitive areas might result from different degrees of palpatory pressure. The method of abdominal palpation followed by the writer was not a deep but a moderate, reasonable pressure such as the condition of the patient would warrant, and the superficial location and the sensitiveness of the parts require. If, however, the results of deep and moderate pressure are at variance, it would be well to have the difference established.

As the result of his personal observations the writer is disposed toward the following conclusions bearing upon the relation of localized tenderness to the site of the causal lesion.

1. The site of the perforated ulcer, as indicated by moderate abdominal palpation within several hours after the onset, is not uniformly *intensely* sensitive.

2. Neither is it always the most sensitive area.

3. In addition to the well recognized local symptoms referable to the region of the perforated ulcer and the appendix, other parts of the abdominal viscera may, exceptionally, be the seat of confusing sensitiveness.

Case 1. D., male, age 50, first seen over twenty-four hours after perforation, refused operation and perished. Autopsy showed diffuse septic peritonitis and perforated duodenal ulcer.

Case 2. P., male, age 35, first seen two hours after perforation, presented only classical symptoms of most intense degree; no diagnostic or operative difficulties. Operation showed perforation of gastric ulcer on the anterior surface to the right of the median lines, close to the greater curvature. Prompt recovery ensued.

Case 3. M., male, age 42 years, was first seen six hours after perforation. The entire abdomen was rigid and retracted. The pyloric region was not specially tender. There was, however, a per-

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2. Keen's Surgery, Vol. III, p. 771 (J. C. Munro).

* Read at the Forty-fourth Annual Meeting of the Medical Society, State of California, Santa Barbara, April, 1914.